Childhood Sodium Chloride Overdose as Culprit in Corruption of Neuronal Behavior in Autism Spectrum Disorder

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Introduction

It has been hypothesized that delayed neuronal response times are to blame for ASD symptoms (as well as certain cognitive advantages,) however, that assumption has always been accompanied by a second assumption that a metabolic i.e. mitochondrial dysfunction underpinned the delayed neuronal response times. While it is, in this author's opinion, a safe bet that delayed neuronal response times are responsible for both the cognitive deficits as well as advantages associated with ASD, the exact cause of those neuronal firing-time delays has been elusive.

Abstract

Autism's meteoric rise in prevalence in the 1980s and 1990s has often been blamed upon vaccines as the number of vaccines administered to children increased during this time in a manner that correlates with the rise in autism and also correlates with the begging of the use of methyl mercury-based preservatives in vaccines in 1990. However, it should be pointed out that the increase in the incidence of autism was already soaring in the 1980s prior to the 1990 introduction of Thimerosal.

Female children account for only 20% of new cases of ASD, a sex-based difference which continues to baffle researchers. My own novel hypothesis seeks to explain this rise in autism, the sex-based difference in ASD risk, as well as how it was that the 1980s and 1990s brought with it this seemingly inexplicable rise in the prevalence of the condition.

A single dietary factor stands out as the most likely responsible factor in this author's mind. That factor is likely to be acute incidences, perhaps a single incident in the case of each patient, of the consumption of large quantities of sodium chloride in any child under the age of 11. To be clear, chronic consumption of sodium chloride is not a cause of ASD, but rather, it is acute incidences (perhaps a single incidence) of excessive NaCl ingestion which I propose is responsible for epigenetic changes which ultimately bring on this condition.

The 1980s brought with it a shift in culture in which children were allowed for the first time to "graze" at will and were given access to salty snacks without parental enforcement of portion control. Whereas previously, parents would serve their children three "square meals" per day and would frequently limit access to food to meal-time, exclusively. The 1980s saw a rise in children being allowed to not merely overeat, but to have direct access to snack foods from the cupboard, many of which tended to be quite salty. Television advertisements exacerbated the problem by targeting children and urging

them to ask their parents for salty snacks which were often handed to their children by the box rather than in small portions.

In the event of an NaCl overdose, neurons seek to protect themselves by implementing semi-permanent epigenetic alterations which instruct proteins responsible for NaCl trans-membrane transport to force nearly all NaCl out of the cytoplasmic medium of select neurons, creating a saline-free or nearly saline-free environment in those neurons. Given that salt autonomously turbulates fluids on on the microscopic level, ions which require transport depend upon the presence of NaCl in order to osmote at a rate capable of supporting rapid neuronal response times. Ideally, there is a moderate quantity of NaCl within healthy neural tissue. The total absence of it in select neurons would give forth the appearance of a mitochondrial disorder similar to Parkinson's Disease, but this is only an appearance. Autism is absolutely not a mitochondrial disorder, but is rather an ion transport disorder with its roots in epigenetic changes brought on by NaCl overdose events which lead ultimately to a condition of zero-salinity in affected neurons.

Conclusion

If there is to be a cure for ASD, researchers will need to devise a method for "flipping back" the genetic switches which are altered by the presence of extreme levels of sodium chloride in victims of the condition. Given that boys are much more likely than girls to overindulge and binge-eat, even at an early age, I propose that this tendency accounts for the sex-based bias in the incidence of the condition.

This hypothesis is further supported by the fact that there have been documented cases of children suddenly losing the ability to speak after having the ability to speak and in rare cases of autism, actually regaining the ability to speak after years of mutism. I propose that these rare cases of an individual losing and then re-gaining the ability to speak are attributable to two distinct incidences of NaCl poisoning spaced apart, resulting in separate clusters of neurons being affected by such epigenetic changes to varying degrees, resulting in two separate response time-delays of varying degrees of severity. In rare cases, when such second incident occurs, function can actually increase as a consequence.